Acute Pancreatitis Secondary to Moderate Hypertriglyceridemia: A Case Report

Anil Nepali[1], Satyam Kharga[1], Malavika Jayan[2], Prakriti Adhikari[3], Amit Shah[1], and Vivek Sanker[4]

[1]Patan Academy of Health Sciences
[2]Bangalore Medical College and Research Institute
[3]Kathmandu University School of Medical Sciences
[4]Government Medical College Thiruvananthapuram

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Anil Nepali[1,2], Satyam Kharga[1], Malavika Jayan[2,3], Prakriti Adhikari[4], Amit Shah[1], Vivek Sanker[2,5]

[1]Patan Academy of Health Sciences,Lalitpur, Nepal

[2]Team Erevnites

[3]Bangalore Medical College and Research Institute, Bangalore, Karnataka, India

[4]Kathmandu University School of Medical Sciences,Dhulikhel, Nepal

[5]Department of Neurosurgery, Trivandrum Medical College, Kerala, India

Corresponding Author:

Name: Dr. Anil Nepali

Affiliation: Patan Academy of Health Sciences, Lalitpur, Nepal

Email: dr.anilnepali1@gmail.com

Abstract

Key Clinical Message

It is crucial to remain vigilant about acute pancreatitis, even in cases with moderately elevated triglycerides. Triglycerides as a cause of acute pancreatitis must be considered even in the absence of other risk factors.

Keywords: *acute pancreatitis, triglyceride, hypertriglyceridemia, Case report* .

Introduction

Hypertriglyceridemia is a common metabolic disorder encountered in clinical practice, characterized by elevated serum triglyceride (TG) levels. The widely accepted normal range of fasting triglyceride is less than 150 mg/dl. (1) (2) When TG levels rise beyond 1000 mg/dL, the risk of acute pancreatitis increases

markedly, making hypertriglyceridemia the third most common cause of acute pancreatitis[1] The complete understanding of how increased triglyceride levels result in acute pancreatitis remains unclear.

It has been hypothesized that the elevated levels of triglyceride-rich lipoproteins interacting with pancreatic lipase in the pancreatic capillaries cause the breakdown of triglycerides into free fatty acids and phospholipids to lysophosphatidylcholine. [3]Additionally, the hydrolysis of triglycerides by lipase induces the generation of toxic levels of free fatty acids and glycerol, thereby damaging the acini and capillaries precipitating edema and hemorrhage.[4] Acute pancreatitis is one of the leading causes of hospitalization among gastrointestinal diseases. Although its diagnosis is simple, the major challenge lies in predicting its progression and outcome. [5]

The clinical diagnosis relies on a combination of history taking, physical examination, and laboratory investigation. Abdominal pain radiating to the back, as well as high serum and urine levels of amylase, point toward acute pancreatitis. [6] The annual incidence of acute pancreatitis varies from 15.9 to 36.4 per 100,000 individuals, with its burden on healthcare resource utilization expected to rise shortly. Despite advancements in healthcare access, imaging tools, and treatments, acute pancreatitis continues to have significant morbidity and mortality. The overall mortality rate varies from 5% to 17% in severe cases and is about 1.5% in mild cases.[7] Herein, we report a rare case of a 26-year-old, para-2, living-2 (P2L2) female with acute pancreatitis caused by moderate triglyceridemia with a triglyceride level of 579 mg/dl.

Case report:

Clinical History:

A 26-year-old, para-2 living-2 (P2L2) female presented to the emergency department of our center with complaints of acute epigastric pain on and off for four days, radiating towards the back and associated with abdominal fullness. She also complained of fever on and off for two days, with a maximum temperature documented at 101.2 degrees Fahrenheit. She had a history of cervical spine surgery with a plate in situ ten years back. She also gave twice a history of lower segment cesarean section (LSCS) and the placement of an intrauterine copper T two years ago after the second cesarean section. She is a non-vegetarian, non-alcoholic, and non-smoker. Her menstrual cycles were regular, each lasting 28-30 days without dysmenorrhea. There was no history of diabetes or pre-diabetes, abdominal trauma, intake of any offending medications, autoimmune diseases, or any other surgical procedures. Her family history was not significant for pancreatitis, dyslipidemia, cardiovascular events, diabetes, gallstones, or autoimmune diseases.

Examination:

On examination, she was tachycardic with a heart rate of 116 beats per minute and normal blood pressure. The rest of her vital signs were within normal limits. She weighed 79 kg, and her height was 157 cm, with a BMI of 32. Her abdominal examination revealed abdominal distention with epigastric tenderness and sluggish bowel sounds.

Investigations:

An ECG was performed, which was found to be normal and ruled out the diagnosis of a cardiovascular accident. Ultrasonography revealed marked hepatomegaly with grade III fatty infiltration, significant free fluid in the peritoneal cavity, copper-T in situ, and an enlarged pancreas measuring 13 cm in long span, with peripancreatic fluid accumulation. The chest X-ray and urine analysis were unremarkable, while the urine pregnancy test was insignificant.

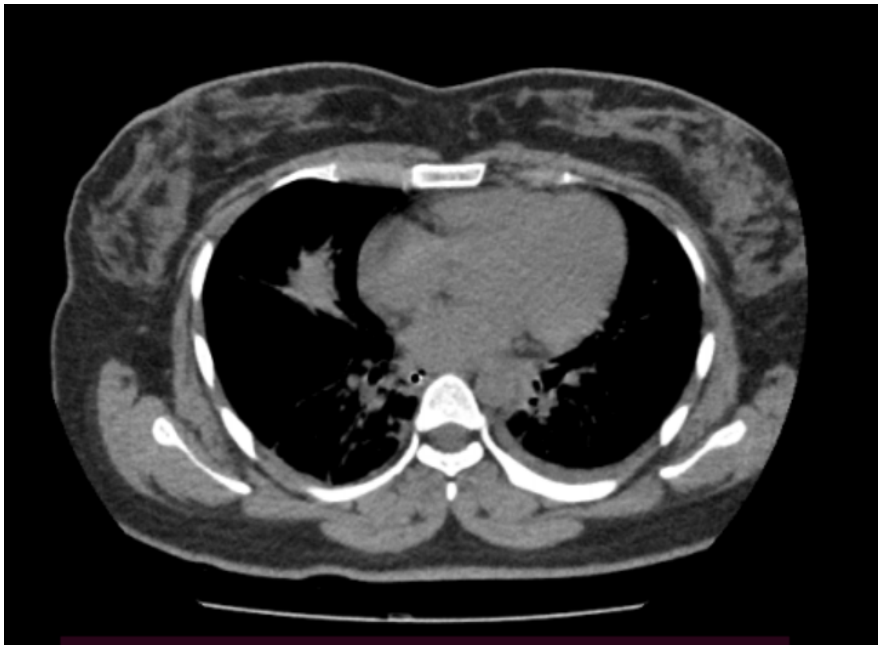
Her laboratory parameters are shown in Table 1.

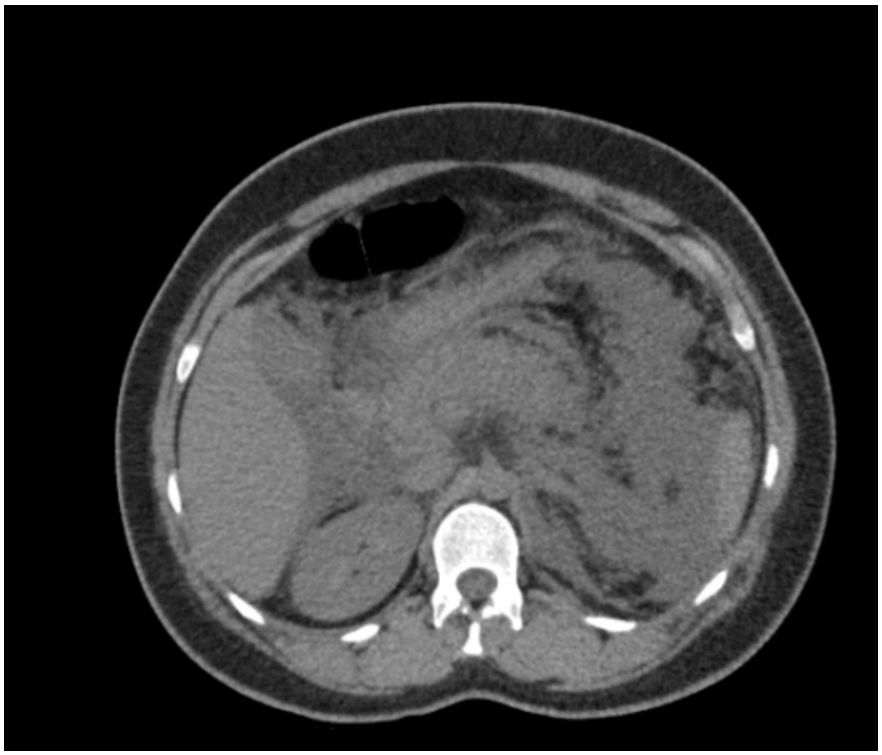
Table

Parameters	**Results at admission**	**Results at 48 hours**

Serum Amylase	451	176
Serum lipase	603	276
Complete blood count	Total count: 16400 Differential count: Neutrophils: 79 Lymphocytes: 18 Monocytes: 3 Eosinophils:0 Basophils:0	Total count: 9500 Differential count: Neutrophils: 62 Lymphocytes:34 Monocytes:4 Eosinophils:0 Basophils:0
Hematocrit (%)	47	35
Serum Hemoglobin (gm/dl)	14.3	11.8
Serum sodium (mmol/L)	133	145
Serum potassium (mmol/L)	4.1	3.6
Serum Urea (mg/dl)	89	46
Serum creatinine (mg/dl)	1.67	0.7
C-reactive protein	52	-
Serum total bilirubin (mg/dl)	2.1	1.7
Serum Albumin (mg/dl)	3.4	3.2
Serum Calcium	8.1	8.5
Corrected Calcium	7.36	9.14
Serum Aspartate aminotransferase	20	14
Serum alanine aminotransferase	50	24
Serum alkaline phosphatase	122	94
Prothrombin time (seconds)	16	13
Serum Total cholesterol	200	188
Serum triglyceride level	579	367
Serum Low-density lipoprotein	72	68
Serum high-density lipoprotein	45	43

A Contrast-Enhanced Computed Tomography (CECT) abdomen was planned; however, a non-contrast CT was done due to low urine output and raised creatinine levels. The non-contract CT scan revealed a diffuse bulky pancreas measuring up to 43 mm at the body, with marked stranding and free fluid accumulation in the peripancreatic region, suggesting acute pancreatitis (Figure 1) with bilateral pleural effusions (Figure 2).





Diagnosis and management:

Based on the clinical findings, such as abdominal pain radiating to the back, elevated triglyceride, serum amylase, and lipase levels, along with positive CT findings, a diagnosis of acute pancreatitis was made. However, challenges arose while looking for the cause of pancreatitis and its severity.

Although the high triglyceride levels were not significant enough to directly cause the symptoms of pancreatitis, considering the absence of various other potential causes like alcohol or drug use, infections, and trauma, we had to explore moderately elevated triglycerides as a possible reason behind the symptoms and went ahead with its targeted treatment. We excluded various potential reasons for the elevated triglyceride levels.

Beginning with secondary factors, she showed no signs of endocrine disorders (treated or untreated thyroid disorder or diabetes), was not under any medication, abstained from alcohol, was not pregnant, and had an unremarkable family history. Additionally, we eliminated possibilities of renal disease, liver disease, and autoimmune disorders. Apart from a high BMI, there were no other identifiable factors contributing to the elevated triglyceride levels. The severity was 2 according to the Bedside Index of Severity in Acute Pancreatitis (BISAP) score and 6 according to the CT severity index (CTSI) score, indicating acute moderate pancreatitis. However, the Ranson score was 1 at the time of admission, indicating acute mild pancreatitis and creating a dilemma in grading the severity of the disease.

The patient was transferred to an intensive care unit and initially managed with aggressive intravenous fluid resuscitation, Fentanyl, and Ondansetron. After collecting samples for blood cultures, empirical antibiotics such as Piperacillin-Tazobactam and Metronidazole were started for high clinical suspicion of infection. Continuous Insulin infusion decreased triglyceride levels to 367 mg/dl on the third day and 223 mg/dl on the seventh day. Fenofibrate 160 mg once daily was started to lower the triglyceride level further and prevent further episodes of pancreatitis. After 48 hours, blood investigations were repeated, which revealed improvement. Empirical antibiotics were stopped as the blood culture reports were insignificant, and the patient was transferred from the ICU to the medical ward. She was discharged on the seventh day of admission, and her vital signs were within normal limits at discharge.

Discussion

High triglyceride levels can stem from primary factors in fewer than 5% of cases, often linked to genetic reasons. More commonly, however, hypertriglyceridemia is secondary to various factors such as diabetes, obesity, pregnancy, excessive carbohydrate intake, hypothyroidism, alcohol consumption, hepatitis, sepsis, renal failure, and specific medications, including estrogen, glucocorticoids, β blockers, bile acid binding resins, thiazides, tamoxifen, cyclosporine, protease inhibitors, and isotretinoin [(8)]. Hypertriglyceridemia is the third most common cause of acute pancreatitis, following alcohol and gallstones, and is classically considered a risk factor only when its levels are higher than 1000 mg/dl. [(1,9)] However, the case described above alerts us to the possibility of acute pancreatitis as a sequela of moderate hypertriglyceridemia.

Pancreatitis secondary to hypertriglyceridemia can sometimes be suspected during physical examination by detecting eruptive xanthomas or lipemia retinalis. Significant increases in triglyceride levels can lead to falsely low serum amylase and lipase, potentially necessitating reliance on pancreatic CT scans for diagnosis. [(3)]

Early management of acute pancreatitis and prevention of its complications are the mainstay of treatment. Initiation of conservative treatment, including aggressive intravenous hydration, initial bowel rest, and pain control, needs to be done soon after the diagnosis is suspected. In addition, several treatment modalities like insulin and heparin, plasmapheresis, combined blood purification therapy (CBPT), High-Volume Hemofiltration (HVHF), and Hemoperfusion (HP) have been described for the targeted treatment of hypertriglyceridemic pancreatitis. [(10)]

An observational study published by Nawaz et al. prospectively enrolled acute pancreatitis patients and categorized them into mild, moderate, and severe based on serum triglyceride levels. The study concluded that elevated serum triglycerides are independently associated with the development of complications like persistent organ failure, regardless of the underlying etiology of acute pancreatitis. Thus, targeting triglyceride-induced lipotoxicity could present an appealing approach for creating new interventions to treat acute pancreatitis. [(11)]

Conclusion

This case highlights the significance of considering moderately elevated triglycerides as a potential cause of acute pancreatitis, emphasizing the necessity for vigilance in diagnosis, especially in the absence of other risk factors. It prompts a re-evaluation of the thresholds for triglyceride levels that might precipitate pancreatitis and emphasizes the need for vigilance, even in moderately elevated triglycerides. More case reports and observational studies are warranted to understand the elevations in triglyceride levels as a causative factor for acute pancreatitis.

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Conflicts of interest:

None declared.

Author contribution:

All the authors contributed equally to drafting, editing, revising, and finalizing the case report.

Ethical approval:

Ethical approval was not required for the case report per the country's guidelines.

Consent:

Written informed consent was obtained from the patient to publish this report.

Data availability statement:

The data supporting this article's findings are available from the corresponding author upon reasonable request.

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